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=> d his

     (FILE 'HOME' ENTERED AT 10:43:45 ON 20 APR 2001)

     FILE 'MEDLINE' ENTERED AT 10:44:45 ON 20 APR 2001
L1             9 S METHYLTRANSFERASE# AND ZINC-FINGER/AB,BI
L2             1 S L1 AND (FUSION OR CHIMER?)/AB,BI

     FILE 'MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS' ENTERED AT 10:45:56 ON
20
     APR 2001
L3            11 S L2
L4             8 DUP REM L3 (3 DUPLICATES REMOVED)
L5           337 S METHYLTRANSFERASE# AND DNA BINDING PROTEIN#/AB,BI
L6            41 S L5 AND (FUSION OR CHIMER?)/AB,BI
L7            30 DUP REM L6 (11 DUPLICATES REMOVED)
L8            22 S L6 AND (TARGET? OR SPECIFIC?)/AB,BI
L9            18 DUP REM L8 (4 DUPLICATES REMOVED)
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L9 ANSWER 16 OF 18 CAPLUS COPYRIGHT 2001 ACS
AN 1992:485813 CAPLUS
DN 117:85813
TI Activation of mammalian DNA **methyltransferase** by cleavage of a zinc binding regulatory domain
AU Bestor, Timothy H.
CS Lab. Hum. Reprod. Reprod. Biol., Harvard Med. Sch., Boston, MA, 02115, USA
SO EMBO J. (1992), 11(7), 2611-17
CODEN: EMJODG; ISSN: 0261-4189
DT Journal
LA English
AB Mammalian DNA (cytosine-5) **methyltransferase** contains a C-terminal domain that is closely related to bacterial cytosine-5 restriction **methyltransferases**. This **methyltransferase** domain is linked to a large N-terminal domain. It is shown here that the N-terminal domain contains a Zn-binding site and that the N- and C-terminal domains can be sepd. by cleavage with trypsin or Staphylococcus aureus protease V8; the protease V8 cleavage site was detd. by Edman degrdn. to lie 10 residues C-terminal of the run of alternating lysyl and glycyl residues which joins the two domains and six residues N-terminal of the first sequence motif conserved between the mammalian and bacterial cytosine **methyltransferases**. While the intact enzyme had little activity on unmethylated DNA substrates, cleavage between the domains caused a large stimulation of the initial velocity of methylation of unmethylated DNA without substantial change in the rate of methylation of hemimethylated DNA. These findings indicate that the N-terminal domain of DNA **methyltransferase** ensures the clonal propagation of methylation patterns through inhibition of the de novo activity of the C-terminal domain. Mammalian DNA **methyltransferase** is likely to have arisen via **fusion** of a prokaryotic-like restriction **methyltransferase** and an unrelated **DNA binding protein**. Stimulation of the de novo activity of DNA **methyltransferase** by proteolytic cleavage in vivo may contribute to the process of ectopic methylation obsd. in the DNA of aging animals, tumors and in lines of cultured cells.